REVIEW ARTICLES

Shaken baby (shaken impact) syndrome: non-accidental head injury in infancy

T J David PhD FRCPCH

J R Soc Med 1999;**92**:556–561 MAC KEITH MEETING 9 JULY 1999

The shaken baby syndrome has been a source of medical controversy for many years, and has been in the public eye since the televised trial of Louise Woodward in Boston in 1997. This paper outlines the condition and discusses some of the more controversial aspects. Is shaking without other trauma sufficient to cause injury? Can injuries from shaking be dated? Can accidents at home or minor trauma cause the condition?

EVOLUTION OF THE TERM SHAKEN BABY SYNDROME

In 1968, Weston, a forensic pathologist in Utah, reported on twenty-one children, aged 1 month to 5 years, with subdural haematoma[1]. All but one had obvious external injuries such as bruising. Injuries resulted from gross violence in each case, such as beating with fists, sticks or other objects, and being thrown or swung against solid objects. In two cases there was mention of violent shaking, in combination with the head being banged against the floor or the cot sides. In 1971, Guthkelch, a neurosurgeon in Hull, pointed out that not all infants with subdural haematoma had external marks of injury on the head, and hypothesized that these infants had been shaken rather than struck[2]. Finally, Caffey, in 1972, suggested that whiplash-shaking was the explanation for cases of subdural haematoma in which there was no sign of external trauma to the scalp[3]; in 1974 he coined the term whiplash shaken infant syndrome[4]. Caffey's hypothesis was that the whiplash shaking might be a component of violence and abuse but might also occur during normal childhood handling such as tossing a baby into the air, 'riding the horse' (baby bouncing on knee of parent), swinging a baby in a circle around the parent or 'skinning the cat' (the child is somersaulted forward while being held by the wrists), or even be caused by noise and vibration from dishwashers, vacuum cleaners and televisions[3].

Belief that shaking is a form of trauma that causes subdural haematoma has led to the name shaken baby syndrome. One source of confusion is that the condition has been given several other labels (Box 1). Another difficulty is that there is no clear definition as to what does or does not constitute the shaken baby syndrome. The term commonly describes a combination of subdural haematoma, retinal haemorrhage and diffuse axonal injury, but there is no consensus on whether it should be applied when violent shaking has damaged the chest wall (e.g. bruising, rib fracture) without causing intracranial injury.

Box 1 **Shaken baby syndrome: other diagnostic labels**

- Shaken impact syndrome
- Whiplash-shaking injury
- Inflicted head trauma
- Abusive head trauma
- Non-accidental head injury

MECHANISMS OF BRAIN DAMAGE IN SHAKEN BABY SYNDROME

Box 2 lists some ways in which the brain may be damaged in shaken baby syndrome; commonly more than one mechanism is in operation.

Box 2 **Mechanisms of damage in shaken baby syndrome**

- Space-occupying effect of large subdural haematoma
- Cerebral oedema
- Cerebral hypoxia
- Contusion of brain
- Tears of the brain
- Diffuse axonal injury

A large subdural haematoma may act as a space-occupying lesion, causing direct compression of the brain and leading to herniation of the brainstem. This, however, is uncommon, and even in fatal cases the quantity of blood in the subdural space may be insufficient to cause a major direct mass effect.

A more usual feature of the syndrome is cerebral oedema, which can be secondary to any of the other

Booth Hall Children's Hospital, Blackley, Manchester M9 7AA, UK
E-mail t.david@netcomuk.co.uk

mechanisms listed in Box 2. If uncontrolled, intracranial pressure can rise to above arterial blood pressure, at which point the brain ceases to be perfused. Cerebral hypoxia may be the consequence of cerebral oedema, but a possible additional mechanism is direct damage to the medulla, with axonal injury (see below) leading to respiratory arrest and hypoxia.

Contusional tears of the brain substance can occur, and are presumed to be the indirect result of either impact or shaking.

Disruption of the axons, known as diffuse axonal injury (DAI), is sometimes a feature. The old concept of DAI was that shearing of axons occurred at the time of injury or very soon after[5]. The new concept is that axons are not torn by shearing or tensile forces at the moment of injury except in extreme circumstances, but instead focal axonal damage impairs axonal transport leading to axonal swelling, disruption and disconnection[6]. DAI is diagnosed by use of histopathological techniques. It is less easy to identify in infants than in older children or adults[7]. Conventional histopathological techniques (e.g. with silver staining) are less sensitive than the newer method of beta-amyloid precursor protein (BAPP) immunostaining. Thus, in one study of twenty-five subjects with fatal head injuries (all but three were adults), DAI was found in eight cases with Palmgren silver staining but in all twenty-five with BAPP staining[6]. Moreover, with conventional techniques DAI may not be detectable until 15 hours after injury. With BAPP staining it can be detected within 2–3 hours of injury; thus in a fatal case a negative result with BAPP staining means either that DAI is not present or that the injury occurred less than 3 hours before death.

Information about the occurrence of DAI in accidental head injuries suggests that impact trauma is the cause, but it has been hypothesized that extreme stretching of the neck, such as might occur in whiplash movements associated with violent shaking, could result in DAI within the medulla, leading promptly to respiratory arrest, cerebral hypoxia and death. If this hypothesis is correct, one would expect to find little in the way of pathological findings other than the non-specific secondary effects of cerebral hypoxia. A potential source of confusion is that BAPP staining is not specific for DAI, and can be the result of cerebral hypoxia[8].

DAI is important because it is probably responsible both for neurological effects (e.g. unconsciousness) immediately after injury and for long-term neurological damage. It is also a cause of cerebral oedema, which in turn is potentially damaging to the brain. Whatever the mechanisms, severe neurological damage and handicap are common outcomes in the shaken baby syndrome[9–11].

BIOMECHANICAL THEORIES

Head injuries result from two types of mechanical force—contact and non-contact. Contact injuries arise when the head strikes or is struck by an object. Non-contact injuries are caused by cranial acceleration or deceleration. Contact forces cause injuries at the site of impact, such as scalp laceration or skull fracture. Contact injuries underlying a skull fracture may include haemorrhage (epidural, subdural or subarachnoid) and parenchymal disruption (contusion, laceration). Contact forces can also cause injuries distant from the impact site, such as basal skull fractures and *contre-coup* parenchymal injuries.

Non-contact strains created by cranial acceleration cause injuries by one of two mechanisms—movement of the brain relative to the skull and dura, with rupture of the bridging veins; and strains within the brain itself, causing DAI[7]. Two types of cranial acceleration are recognized, separately or in combination. *Translational* acceleration occurs when the brain's centre of gravity, approximately the pineal gland, moves in a straight line; *rotational* or *angular* acceleration occurs when the head turns about its centre of gravity (without linear movement of the centre of gravity)[7]. Experimental work suggests that the duration of cranial acceleration is a factor: in animals concussion was more likely with a long duration of acceleration that allowed time for deep brain parenchymal strains; with shorter duration, greater acceleration was required to produce loss of consciousness[12].

Much of the experimental information comes from studies in which monkeys, fixed into a rigid seat, were fitted with a helmet that was jolted or vibrated[13,14]. Another experimental model has been an infant-sized doll with its head packed tight with wet cotton waste and fitted with an accelerometer[14]. The doll was vigorously shaken, to generate forces of the kind known to cause concussion, subdural haematoma or DAI. The overall conclusion was that most, if not all, severe inflicted injuries are due to sudden deceleration associated with the forceful striking of the head against a surface[15]. By contrast, the main cause of the subdural haematomas and DAI seen in motor accident victims is believed to be forceful rotation of the brain about its centre of gravity. Biomechanical studies have suggested that when the head of an infant model held by the trunk strikes a surface the angular acceleration is fifty times greater than when the head is merely shaken, and the injury thresholds calculated for infants are not reached until the moment of impact[15]. It is the sudden angular deceleration experienced by the brain and cerebral vessels, not the specific contact force applied to the surface of the head, that results in the intracranial injury[15].

Numerous differences between these biomechanical models and the living human infant have raised questions about the need to invoke impact, over and above shaking, to explain the tearing of the bridging veins (which leads to subdural haematoma), retinal haemorrhage (not seen in most cases of accidental head injury) and DAI[16]; but most infants with abusive head injuries have clinical, radiological, or post-mortem evidence of blunt impact to the head, and the term shaken-impact syndrome probably reflects more accurately than shaken baby syndrome the usual mechanism responsible for these injuries.

The current synthesis of all this information is that infants with abusive head trauma have commonly been subjected to several mechanisms of injury—impact trauma, short-duration head acceleration in the sagittal plane producing subdural haemorrhage and long-duration cranial acceleration in the coronal plane producing DAI[7,17].

CAN SHAKING WITHOUT OTHER TRAUMA CAUSE NON-ACCIDENTAL HEAD INJURY?

There is no proof of the shaking hypothesis. Various types of child abuse have been observed during covert video surveillance but there are no published records of shaking leading to subdural and retinal haemorrhage. Several forensic pathologists in the UK and the USA have expressed doubt that shaking alone can cause subdural haematoma or other intracranial injury[18–22]. They and others have pointed to the rarity with which a history of shaking is obtained in affected infants.

One observation that was initially believed to favour the shaking hypothesis was the absence of scalp injury in some cases of subdural haematoma. The implication was that impact injury could not be the cause, leaving shaking as the only remaining candidate. However, the likelihood of scalp trauma will be determined by the type of surface with which the head makes contact: a child thrown against a soft cot mattress, bed, or carpeted floor can sustain severe brain injuries without evident external damage[23].

In favour of the shaking hypothesis are confessions of violent shaking[3]. But the difficulty with confessions, other than their well-documented unreliability[24,25], is that potential perpetrators may come under duress to confess to shaking when it has not occurred. A parent may feel pressurized to confess by professionals who believe (and the belief is widely held) that an abused child cannot safely be returned to a perpetrator *without* a confession. A further factor is that someone who has caused a head injury by an act of gross violence, such as slamming the baby's head onto a bed, may be tempted to admit to the seemingly less serious act of shaking. For these reasons, when interviewing carers or suspect perpetrators, doctors and others have been advised not to mention shaking as a mechanism of injury[26]. Not all confessions, of course, are false.

Although often not present, the strongest indicators that a child has been subject to violent and abusive shaking are finger or hand mark bruising of the chest wall or arms, sometimes coupled with fractures of the rib cage. Although such injuries strongly suggest violent shaking, this does not necessarily prove that shaking (rather than impact) was the cause of the head injury.

Retinal haemorrhages are uncommon in severe *accidental* impact head trauma. It is therefore hard to attribute the high incidence of retinal haemorrhage in infants with subdural haematoma to impact trauma, and violent shaking is the most plausible explanation.

CAN THE SHAKEN BABY SYNDROME RESULT FROM MINOR TRAUMA?

The mechanical properties of normal living human tissues afford infants and young children considerable protection. Several studies have shown that skull fractures are an infrequent complication of witnessed indoor accidental falls, and children can survive falls from great heights. There are no data to support Caffey's hypothesis of major injury as a result of normal childhood handling, or of vibration and noise from domestic appliances, and it seems that major trauma is usually required to produce subdural haematoma and retinal haemorrhage. There are, however, exceptions; for example, in three reported cases of subdural haematoma and retinal haemorrhage there had been minor unintentional household trauma such as falling less than one metre onto the floor[27], and a less well documented case occurred after a stairway fall[28]. Hypernatraemia used to be regarded as a non-traumatic cause of subdural haematoma, but a forthcoming report casts great doubt on this idea[29].

RE-BLEEDING OF AN OLD SUBDURAL HAEMORRHAGE AFTER MINIMAL TRAUMA?

Re-bleeds are believed to occur frequently in resolving subdural haematomas, but the amount of bleeding around the existing haematoma is seldom large[26]. There is no evidence that re-bleeding can cause serious brain injury[26]. The hypothesis is sometimes advanced that the existence of an old, small, chronic subdural haematoma or effusion can predispose to the development of massive and life-threatening acute subdural haematoma as the result of very minor trauma or normal handling. One mechanism that has been suggested for this putative phenomenon is that excess extracerebral fluid allows the brain more freedom to move about within the cranium, thereby rendering the bridging veins more prone to

tearing. If this mechanism did indeed apply, one would expect to see subdural haematoma in association with other causes of excess extracerebral cerebrospinal fluid, such as post-meningitis subdural effusion, communicating hydrocephalus or cerebral atrophy, but with rare exceptions (some of which have been reported on the internet) infants with these conditions have not developed subdural haematoma.

THE LOCATION OF INTRAOCULAR HAEMORRHAGES IN THE SHAKEN BABY SYNDROME

Haemorrhages may lie in front of the retina (preretinal), within the layers of the retina (intraretinal) or beneath the retina (subretinal)[30]. Subretinal haemorrhages are distinguished by the presence of overlying blood vessels. Intraretinal haemorrhages are often described as dot, blot or flame. Size distinguishes dot from blot haemorrhages; both are roughly circular. Flame haemorrhages lie within the nerve fibre layer, which is superficial, and blood in this layer follows the nerve fibres causing the edges to have a feathered or flame-shaped appearance. Flame, dot and blot haemorrhages will tend to cause blood vessels to 'disappear' as they run below or through the blood. Blood in the potential space between the vitreous and the retina is known as subhyaloid haemorrhage. Haemorrhage may be found within the optic nerve sheath, and is typically most obvious at the junction of the nerve and the globe[15].

Retinal haemorrhages tend to be small, few in number, flame-shaped and located on and radiating around the optic nerve. These haemorrhages are not caused by trauma *per se*, and can be seen in any case of papilloedema[30].

The appearance of the retina in the shaken baby syndrome may range from normal, through a few dot or blot haemorrhages in the posterior pole, to extensive almost confluent intraretinal haemorrhage covering the entire retinal surface out to the ora serrata[30]. In the most severe cases, vitreous haemorrhage, traumatic retinoschisis (retinal splitting), perimacular retinal folds and retinal detachment may be present[27]. A correlation has been reported between the severities of retinal and cerebral injuries in non-accidental trauma[31,32]. The findings in the eyes may be asymmetrical or even unilateral.

THE PATHOGENESIS AND SIGNIFICANCE OF RETINAL HAEMORRHAGES

Childhood conditions that have been documented to be associated with retinal haemorrhage are listed in Box 3. In many the association with retinal haemorrhage is weak, if not frankly tenuous, and most conditions have distinguishing clinical features.

Box 3 **Conditions of childhood that can be associated with retinal haemorrhage**

- Hereditary bleeding disorders, e.g. haemophilia, von Willebrand disease
- Leukaemia
- Haemorrhagic disease of the newborn
- Retinopathy of prematurity
- Sickle cell retinopathy
- Extracorporeal membrane oxygenation treatment
- Galactosaemia
- Henoch–Schönlein purpura
- Intraocular surgery
- Severe hypertension
- Maternal ingestion of cocaine
- Meningococcal meningitis
- Severe chronic papilloedema
- Optic disc drusen
- X-linked retinoschisis
- Intracranial vascular malformations
- Tuberous sclerosis
- Convulsions

Retinal haemorrhages are common at birth; most disappear by eight days[33–35] but some persist for up to three months[36]. Retinal haemorrhage can occur as a result of accidental injury, but only rarely. In studies of one hundred and forty-eight children under 3 years of age with accidental head injury, including fifty-eight with skull fracture and forty with falls from higher than 1.2 m, retinal haemorrhage was found in only one child, the victim of a high-speed car accident[37,38]. In contrast, retinal haemorrhages were found in twelve of twenty-seven suspected abuse cases.

The pathogenesis of intraocular haemorrhage in the shaken baby syndrome is poorly understood. Two main mechanisms have been proposed. One is a shearing effect on the retina at the junction of the vitreous to the retina, particularly at areas of rigid adherence such as around the optic nerve and at the pars plana. A shearing mechanism is supported by one report of the distribution of haemorrhages[31], and the shearing forces from vitreous shaking would also explain traumatic retinoschisis. The other suggested mechanism is a rise in intraocular venous pressure, due to a rise in intracranial pressure, an increase in central venous pressure or rupture of vessels within the subarachnoid space[39]. All result in pressure on the central retinal vein[39]. It is increasingly the view that retinal haemorrhage in the shaken baby syndrome is most likely to be the direct result of shaking of the globe, though the precise mechanism by which this could produce intraocular haemorrhage is unclear[30].

Cardiopulmonary resuscitation has been advanced as a possible cause of retinal haemorrhage[40–42], but studies of children who have been resuscitated indicate that such resuscitation alone is very unlikely to cause retinal haemorrhage, even if done by unskilled individuals[36].

AGEING OR DATING OF INJURY

Objective data are lacking on the time that elapses between injury and onset of symptoms, and there are only a small number of well-documented case histories[16,43]. Information about the timing of the injury must be extrapolated from data on accidental trauma[44]. Acute subdural haematoma associated with severe neurological compromise, brain swelling, or death occurs in the setting of a clear injury involving a major mechanical force and is followed by the immediate or rapid onset of neurological symptoms[15]. In a series of ninety-five children who died from accidental head injuries, all but one had an immediate decrease in the level of consciousness; the exception was a patient with an expanding epidural haematoma, a type of injury not usually associated with child abuse[44]. On the basis of these data, one can conclude that a child with a fatal shaken-impact injury will develop obvious neurological symptoms very rapidly.

The timing of the traumatic event is far more difficult, if not impossible, to establish in those with less severe neurological injuries, in whom the symptoms are much more variable. Some injuries, especially in infants, are occult[45]. The timing of injury is particularly hard to estimate in children with delayed intracerebral haematoma—a condition not generally associated with shaken-impact injury in which sudden neurological deterioration follows a period of mild symptoms[46–48].

Fractures of long bones can be dated radiologically. However, skull fractures, for reasons that are obscure, do not produce callus during healing, and cannot be dated radiologically. Evidence of healing can be seen histologically, so some information on age may be obtainable at necropsy.

Skin bruises change colour with time and can be approximately dated, but in the eye the colour changes that occur in skin bruising are absent[36]. Accurate dating of retinal haemorrhages, based on their ophthalmoscopic appearances, is not possible. Superficial flame-shaped intraretinal haemorrhages clear within days, but large intraretinal haemorrhages, dome-shaped haemorrhages or prehyaloid haemorrhages may take months to clear[36]. The persistence of haemosiderin, the result of bleeding at least 2 days previously, is poorly documented.

Some crude information about timing may be obtained from the computed tomographic or magnetic resonance appearance of the blood in a subdural haematoma. Further information about timing may be obtained at necropsy. A particular area of uncertainty is the time taken for disappearance of haemorrhage resulting from birth trauma.

CONCLUSIONS

The great majority of infants with the triad of subdural haematoma, retinal haemorrhage and DIA have been exposed to gross non-accidental trauma. The relative importance of shaking and impact remains uncertain. Overlap with accidental trauma and injuries occurring at birth is an area of diagnostic difficulty in some cases. Non-accidental head injury is commonly associated with a poor neurological outcome, and damage to the eyes and optic nerves is responsible for permanent visual handicap in some cases.

Acknowledgments This review arose in part from presentations at an RSM MacKeith meeting entitled 'Non-accidental head injury in infants—"shaken baby syndrome"' on 9 July 1999, at which the speakers were Dr J Livingstone, Dr R A Minns, Dr A Parsons, Dr T Jaspan, Dr M McPhillips, Dr P Andrews, Dr K M Barlow, Mr J Punt, Professor J Bell, Dr J K Brown and Dr J Mok. I thank Professor Helen Carty, Mr Chris Lloyd and Dr Robert Sunderland for their comments.

REFERENCES

1 Weston JT. The pathology of child abuse. In: Heifer RE, Kempe CH, eds. *The Battered Child*. Chicago: University of Chicago Press, 1968: 77–100

2 Guthkelch AN. Infantile subdural haematoma and its relationship to whiplash injuries. *BMJ* 1971;**2**:430–1

3 Caffey J. On the theory and practice of shaking infants. Its potential residual effects of permanent brain damage and mental retardation. *Am J Dis Child* 1972;**124**:161–9

4 Caffey J. The whiplash shaken infant syndrome: manual shaking by the extremities with whiplash-induced intracranial and intraocular bleedings, linked with residual permanent brain damage and mental retardation. *Pediatrices* 1974;**54**:396–403

5 Maxwell WL, Watt C, Graham DI, Gennarelli TA. Ultrastructural evidence of axonal shearing as a result of lateral acceleration of the head in non-human primates. *Acta Neuropathol* 1993;**86**:136–44

6 Gentleman SM, Roberts GW, Gennarelli TA, *et al*. Axonal injury: a universal consequence of fatal closed head injury? *Acta Neuropathol* 1995;**89**:537–43

7 Hymel KP, Bandak FA, Partington MD, Winston KR. Abusive head trauma? A biomechanics-based approach. *Child Maltreat* 1998;**3**:116–28

8 Shannon P, Smith CR, Deck J, Ang LC, Ho M, Becker L. Axonal injury and the neuropathology of shaken baby syndrome. *Acta Neuropathol* 1998;**95**:625–31

9 Bonnier C, Nassogne MC, Evrard P. Outcome and prognosis of whiplash shaken infant syndrome; late consequences after a symptom-free interval. *Dev Med Child Neurol* 1995;**37**:943–56

10 Ewing-Cobbs L, Kramer L, Prasad M, *et al*. Neuroimaging, physical, and developmental findings after inflicted and noninflicted traumatic brain injury in young children. *Pediatrics* 1998;**102**:300–7

11 Sinal SH, Ball MR. Head trauma due to child abuse: serial computerized tomography in diagnosis and management. *South Med J* 1987;**80**:1505–12

12 Ommaya AK, Faas F, Yarnell P. Whiplash injury and brain damage. *JAMA* 1968;**204**:75–9

13 Hanigan WC, Peterson RA, Njus G. Tin ear syndrome: rotational acceleration in pediatric head injuries. *Pediatrics* 1987;**80**:618–22

14 Duhaime AC, Gennarelli TA, Thibault LE, Bruce DA, Margulies SS, Wiser R. The shaken baby syndrome. A clinical, pathological, and biomechanical study. *J Neurosurg* 1987;**66**:409–15

15 Duhaime AC, Christian CW, Rorke LB, Zimmerman RA. Nonaccidental head injury in infants—the "shaken-baby syndrome". *N Engl J Med* 1998;**338**:1822–9

16 Sunderland R. Skull fracture, subdural haematoma, shaking and impact injury. In: David TJ, ed. *Recent Advances in Paediatrics*. 17th edn, Edinburgh: Churchill Livingstone, 1999:35–56

17 Gennarelli TA, Thibault LE. Biomechanics of acute subdural hematoma. *J Trauma* 1989;**22**:680–6

18 Knight B. Fatal child abuse. In: *Forensic Pathology*, 2nd edn, New York: Oxford University Press, 1996:466–8

19 Leadbeatter S, James R, Claydon S, Knight B. The shaken infant syndrome. Shaking alone may not be responsible for damage. *BMJ* 1995;**310**:1600

20 Plunkett J. Shaken baby syndrome and the death of Matthew Eappen. A forensic pathologist's response. *Am J Forensic Med Pathol* 1999;**20**:17–21

21 Di Maio VJM, Dana SE. *Handbook of Forensic Pathology*. Austin, Texas: Landes Bioscience, 1998

22 Norton LE. Child abuse. *Clin Lab Med* 1983;**3**:321–42

23 Monteleone JA, Brodeur AE. *Child Maltreatment. A Clinical Guide and Reference*. St Louis: G W Medical, 1994

24 Gudjonsson G. *The Psychology of Interrogations, Confessions and Testimony*. Chichester: John Wiley, 1998

25 Gudjonsson GH, Haward LRC. *Forensic Psychology. A Guide to Practice*. London: Routledge, 1998

26 Showers J. Never never never shake a baby. The challenge of the shaken baby syndrome. *Proceedings of the Second National Conference on Shaken Baby Syndrome, September 1998, Salt Lake City, Utah*. Alexandria, Virginia: National Association of Children's Hospitals and Related Institutions, 1999

27 Christian CW, Taylor AA, Hertle RW, Duhaime AC. Retinal hemorrhages caused by accidental household trauma. *J Pediatr* 1999;**135**:125–7

28 Chiaviello CT, Christoph RA, Bond GR. Stairway-related injuries in children. *Pediatrics* 1994;**94**:679–81

29 Handy TC, Hanzlick R, Shields BA, Reichard R, Goudy S. Hypernatremia and subdural hematoma in the pediatric age group: is there a relationship? *J Forensic Sci* (in press)

30 Levin AV. Intraocular manifestations of the shaken baby syndrome: retinal haemorrhages and child abuse. In: David TJ, ed. *Recent Advances in Paediatrics*, 18th edn, Edinburgh: Churchill Livingstone (in press)

31 Green MA, Lieberman G, Milroy CM, Parsons MA. Ocular and cerebral trauma in non-accidental injury in infancy: underlying mechanisms and implications for paediatric practice. *Br J Ophthalmol* 1996;**80**:282–7

32 Wilkinson WS, Han DP, Rappley MD, Owings CL. Retinal hemorrhage predicts neurological injury in the shaken baby syndrome. *Arch Ophthalmol* 1989;**107**:1472–4

33 Kaur B, Taylor D. Retinal haemorrhages. *Arch Dis Child* 1990;**65**:1369–72

34 Planten JT, Van der Schaaf PC. Retinal haemorrhage in the newborn. An attempt to indicate and explain its cause and significance. *Ophthalmologica* 1971;**162**:213–22

35 Baum JD, Bulpitt CJ. Retinal and conjunctival haemorrhage in the newborn. *Arch Dis Child* 1970;**45**:344–9

36 Taylor D, Bonshek R, Brosnahan D, *et al*. Child abuse and the eye. *Eye* 1999;**13**:3–10

37 Buys YM, Levin AV, Enzenauer RW, *et al*. Retinal findings after head trauma in infants and young children. *Ophthalmology* 1992;**99**:1718–23

38 Duhaime AC, Alario AJ, Lewander WJ, *et al*. Head injury in very young children: mechanisms, injury types, and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. *Pediatrics* 1992;**90**:179–85

39 Good WV. Accidental and non-accidental trauma. In: Taylor D, ed. *Paediatric Ophthalmology*, 2nd edn, Oxford: Blackwell Science, 1997:840–66

40 Bacon CJ, Sayer GC, Howe JW. Extensive retinal haemorrhages in infancy—an innocent cause. *BMJ* 1978;**2**:281

41 Goetting MG, Sowa B. Retinal hemorrhage after cardiopulmonary resuscitation in children: an etiologic reevaluation. *Pediatrics* 1990;**85**:585–8

42 Weedn VW, Mansour AM, Nichols MM. Retinal hemorrhage in an infant after cardiopulmonary resuscitation. *Am J Forensic Med Pathol* 1990;**11**:79–82

43 Nashelsky MB, Dix JD. The time interval between lethal infant shaking and onset of symptoms. A review of the shaken baby syndrome literature. *Am J Forensic Med Pathol* 1995;**16**:154–7

44 Willman KY, Bank DE, Senac M, Chadwick DL. Restricting the time of injury in fatal inflicted head injuries. *Child Abuse Negl* 1997;**21**:929–40

45 Greenes DS, Schutzman SA. Occult intracranial injury in infants. *Ann Emerg Med* 1998;**32**:680–6

46 Morin MA, Pitts FW. Delayed apoplexy following head injury ("traumatische Spät-Apoplexie"). *J Neurosurg* 1970;**33**:542–7

47 Young HA, Gleave JRW, Schmidek HH, Gregory S. Delayed traumatic intracerebral hematoma: report of 15 cases operatively treated. *Neurosurgery* 1984;**14**:22–5

48 Solonuik D, Pitts LH, Lovely M, Bartkowski H. Traumatic intracerebral hematomas: timing of appearance and indications for operative removal. *J Trauma* 1986;**26**:787–93